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Co-occuring Toxoplasma Infection and Psychotic Symptoms : Case Report

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Abstract

Introduction

Schizophrenia is a neuropsychiatric disease that is global and is experienced by 1% of the population in the United States and Europe. This study raises awareness of the role of infectious agents in the initiation of psychotic symptoms in schizophrenia.

Case Presentation

A 20-year-old man is taken by the family to the emergency department because he has decreased consciousness, and the body suddenly stiffens. Patients begin to experience changes in behavior in the form of difficulty sleeping, when invited to talk quietly, laughing alone and whispering without the other person. TORCH examination found an increase in anti-toxoplasma IgM and IgG. This patient is then given basic life support in the form of ABC (airway, breathing, circulation support) and seizure management. Also given risperidone 2 mg / 12 hours, pyrimethamine 1-II (1x200 mg), pyrimethamine day III-XXI (1x 25 mg), intravenous Cefotaxim 2g / 8 hours, Clindamycin 500 mg / 8 hours. The patient experienced improvement after the second week of treatment.

Conclusion

Toxoplasmosis causes lesions in the brain that cause changes in brain neurotransmitter pathways, which lead to changes in patient behavior.

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Keywords: Toxoplasmosis, Psychotic, Schizophrenia

Introduction

Schizophrenia is a neuropsychiatric disease that is global and is experienced by 1% of the population in the United States and Europe. An increased risk of developing

schizophrenia in individuals with a history of family members with the same mental illness indicates that the disease has a genetic or hereditary etiology. Environmental factors also determine the emergence of schizophrenia. Epidemiological studies, for example, have established that winter-spring births, city births, and perinatal and postnatal infections are all risk factors that influence the development of schizophrenia in a person. These studies have re-increased interest in the role of infectious agents in schizophrenia. A study states there is a relationship between toxoplasma infection with the appearance of psychotic symptoms resembling schizophrenia.[1]

T. gondii is the most intracellular parasite found in toxoplasmosis. Its life cycle can only be completed in cats and rodents, which are the definitive hosts. However, T. gondii also infects a variety of intermediary hosts, including humans. Toxoplasma organisms have also been shown to interfere with learning and memory in mice and produce behavioral changes in mice and rats. What's interesting is the research that shows that mice infected with Toxoplasma become less neophobia, which causes a reduction in their natural aversion to the smell of cats. This change in behavior increases the likelihood that mice will be eaten by cats, thus allowing Toxoplasma to complete its life cycle, an example of manipulation of host behavior driven by parasites by parasites. Some cases of acute toxoplasmosis in adults are associated with psychiatric symptoms such as delusions and hallucinations.[2-4] This case report will describe a toxoplasma infection accompanied by psychotic symptoms in our patients.

Case Presentation

A man aged 20 years, a student, Javanese, Catholic, living in a village in Bantul, Yogyakarta, was brought by his family to the emergency department at Sardjito Hospital, Yogyakarta.

Based on alloanamnesis, we found prenatal history as follows; the patient was an unwanted baby, born when the mother was 20 years old. The situation of the mother during pregnancy is quite depressed because the patient is a pregnant child out of wedlock. The patient's mother was not approved to marry the patient's biological father

because of religious differences. The patient was cared for by her mother until the age of 2 years. After 2 years, patient care was left to grandma because the mother would work in Jakarta. According to the family, the achievement of patients in school is quite good, patients were classified as smart children in school. Somehow, there were some bad experiences that happened when he was a child. During elementary school the patient once asked his teacher about who his father was. However, the teacher answered his father was absent. Then the patient asked the same thing to grandfather, then was answered that his father was the patient's grandfather. Patients also often feel ashamed because they are often bullied by their friends.

This patient was taken by the family to the emergency department because of decreased consciousness, and the body suddenly stiffened. . 4 days before entering the hospital, the patient began experiencing changes in behavior in the form of insomnia, when invited to talk quietly, laughing alone and whispering without any interlocutors. The patient is then taken by the family to the priest, but there is no improvement. 1 day before entering the hospital, the patient went berserk against the family, did not want to sleep, eat-drink and take care of themselves. The patient laughs more often and does not want to answer if asked one month before entering the hospital, the patient suddenly stiffened in the legs and arms. According to friends, at that time the patient was unconscious during the seizure, and after the seizure regained consciousness. CT scan results are normal. The patient had not previously been treated for psychiatric disorders.

Physical examination and neurologic status are within normal limits. There was an increase in SGOT (84 u / l), Glucose during (150 mg / dl), Neutrophil count (81%) Lymphocytes (10.5%). Urinalysis showed an increase in erythrocyte count (13,207), an increase in leukocyte count (7770), and bacteria were found in the urine. TORCH examination found an increase in anti-toxoplasma IgM and IgG.

This patient is then given basic life support in the form of ABC (airway, breathing, circulation support) and seizure management. Also given risperidone 2 mg / 12 hours, pyrimethamine 1-II (1x200 mg), pyrimethamine day III-XXI (1x 25 mg), intravenous Cefotaxim 2g / 8 hours, Clindamycin 500 mg / 8 hours.

The patient experienced improvement after the second week of treatment. The patient begins to regain consciousness and begin to be responsive when asked. The patient begins to be able to answer a number of questions, although it still seems weak. In psychiatric interviews, autistic thinking, blocking, hypothimic mood, and auditory halucination were obtained.

Psychotherapy is given to families who care for patients, including supportive psychotherapy and education about drug compliance after discharge from hospital. After about 1 month of treatment, the patient experiences partial recovery, although some sequelae such as blocking and hearing hallucinations are sometimes still experienced.

Discussion

Toxoplasmosis is an infection caused by Toxoplasma gondii, an intracellular parasitic protozoan (non-flagellate organism), which infects most warm-blooded animals, including humans. Toxoplasmosis has three stages of infection (infective stages), namely rapidly dividing invasive tachizoite, slowly dividing bradyzoite in tissue cysts, and environmental stage sporozoite, which is protected in oocysts. The prevalence of toxoplasmosis in the world's population is around 25 to 30% with variations between countries 10-80%. Low seroprevalence (10-30%) was observed in North America, Southeast Asia and Sahelia countries in Africa. Moderate prevalence (30-50%) is found in Southern and Central Europe, and high prevalence is found in Latin America and tropical African countries.[5-7]

T. gondii is considered a zoonosis worldwide. Infection in humans occurs through consumption of cysts which are often found in raw or undercooked meat, oocysts present in contaminated water or other food (vegetables) or by default through transplacental transmission of tachyzoites if the mother suffers from acute infection during pregnancy. Other forms of transmission have been described including transplantation of infected organs or transfusion of contaminated blood cells.[8,9]

The life cycle of T. gondii is complex and may involve many hosts. Felines (domestic cats) are considered as the definitive hosts and humans and various other warm-blooded animals function as intermediary hosts. Both types of host infections occur through ingestion of tissue cysts. Intermediary hosts can also be infected through ingestion of oocysts and tachyzoites.[10,11]

Once ingested, the cyst will release slow multiplier bradyzozoites in the intestinal tract, which will attack epithelial cells and become tachyzoites that multiply very quickly, and will continue to replicate and infect any nucleated cells until the immune response is mediated by these pressing cells. replication that results in chronic infection with the formation of tissue cysts. The parasitic sexual stages will only occur in definitive host intestinal epithelial cells. This will cause shedding of oocysts in the stool, which will then sporulate and become infective after contact with the atmosphere.[12,13]

In intermediate hosts, tissue cysts can survive indefinitely in the brain or muscles. However, when asymptomatic individuals exhibit some immune deficiency, reactivation of the latent form can occur, resulting in severe tissue damage. As cysts (filled with bradyzoites) have a tendency for nerve cells, eyes and muscles, reactivation (tachyzoites) will often be present as chorioretinitis, myalgia and cerebral toxoplasmosis, which are usually fatal if not treated.[11-13]

Psychosis syndrome occurs associated with increased dopamine neurotransmitter activity (hyperactivity of the central dopaminergic system). The action of a typical antipsychotic drug is to block dopamine at post synaptic receptors on neurons in the brain, especially in the limbic system and the extrapyramidal system (dopamine D2 receptor antagonist), so it is effective for positive symptoms. While the new (atypical) antipsychotic drug besides having an affinity for dopamine D2 receptors is also against 5HT serotonin receptors (serotonin dopamine antagonists), so that it can stabilize dopamine in the mesocortex pathway so that it is effective for negative symptoms.[14]

Pyrimethamine is a folic acid antagonist that is used as an antimalarial or with sulfonamides to treat toxoplasmosis. The choice of pyrimethamine as a regimen for treating toxoplasmosis is due to the specific therapeutic action of distinguishing between host and parasite based on nucleic acid precursors. The effectiveness of pyrimethamine in treating toxoplasma gondii increases when combined with sulfonamides.[15]

Folic acid is a derivative of 5 formyl tetrahydrofolic acid. Folic acid plays a role in purine / pyrimidine synthesis in the inhibition of dihydrofolate reductase, so that the process of DNA replication and RNA transcription can take place.[15]

Clindamycin is effective in inhibiting bacterial growth, by blocking the dissociation of peptidyl transfer RNA (t-RNA) from the ribosome, resulting in the termination of RNA-dependent protein synthesis. Clindamycin is an alternative to sulfonamides. Clindamycin is beneficial when used in conjunction with pyrimethamine in the treatment of acute toxoplasmosis in the central nervous system that occurs in patients with HIV-AIDS.[16]

Conclusion

Toxoplasmosis causes lesions in the brain that cause changes in brain neurotransmitter pathways, which lead to changes in patient behavior.

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